Fungal Infection Study Forum

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COROLLARY TO OCCAM'S RAZOR

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A 45 year old gentleman from Chennai, who had undergone deceased donor renal transplant in November 2018 and had a reasonably stable graft function on triple immunosuppression with Tacrolimus, Mycophenolate & Prednisolone and Cotrimoxazole prophylaxis. He presented in February 2019 with a lower respiratory tract infection with pleuritic pain which was treated with antibiotics by the Nephrology team. Since his symptoms did not get better, he was referred to the ID team for further evaluation. His past medical history was significant in that he had received empirical Antitubercular treatment 7 years back and had undergone pancreatic duct stenting in October 2018 (1 month before the transplant). Clinical examination revealed a low grade fever with stable hemodynamics and a room air saturation of 95 %. On systemic examination of the respiratory system scattered crepitations were heard.

With this clinical background, patient was further subjected to radiological assessment in the form of chest skiagram and CT chest which showed bilateral multiple nodules with surrounding ground glass opacity noted around few nodules, with predominant subpleural distribution with mosaic attenuation and air trapping (Figure 1). Blood cultures were negative. While waiting on fungal biomarkers (Beta D glucan, serum cryptococcal antigen), bronchoscopy was done. BAL GeneXPert Ultra for M.TB Rif was negative and so was bacterial culture. BAL galactomannan was 2.7. Aerobic cultures were sterile. Fungal culture grew Aspergillus flavus and fumigatus. A transthoracic biopsy was also done to establish a definitive diagnosis of invasive fungal infection. Histopathological examination was suggestive of a mixed invasive fungal infection with aseptate broad fungal hyphae and septate fungal hyphae in a necrotic background. Biopsy cultures also grew Aspergillus Screening brain imaging did not reveal any intracranial lesion.

Patient was started on Inj. Amphotericin B and later switched over to oral Posaconazole. He tolerated therapy well and serum Posaconazole level was in the therapeutic range 1.6 mcg/ml. Immunosuppressive therapy was reduced and monitored by the primary team. His symptoms got better and follow up CT chest after 3 weeks showed significant reduction in size of most of the pulmonary nodules (Figure 2).

While on Posaconazole, 2 months later, patient developed neurological symptoms (left sided hemiparesis). MRI Brain with contrast revealed a Space occupying lesion with perilesional edema in the right parietal region. With a background of mixed mycotic infection in the lungs, attributing neurological worsening to metastatic fungal etiology would be logical. Craniotomy was done and pus from the brain abscess revealed gram positive thin filamentous bacteria suggestive of Nocardia and 16s from the sample was reported as *Nocardia farcinica*. On retrospective

Message from the Editor

Dear All,

On behalf of the FISF, it gives me great pleasure in welcoming the delegates of the 32nd Annual Conference of the Indian Society of Organ Transplantation at Nagpur.

This newsletter exclusively discusses fungal infections in recipients of solid organ transplants. We have a pot pourri of cases including invasive candidiasis, invasive aspergillosis. mucormycosis, cryptococcosis, histoplasmosis and dematiaceous fungi. The cases illustrate the diverse diagnostic and therapeutic approaches in all these patients. They particularly highlight the importance of invasive methods for diagnosis, proper interpretation of reports, source control and attention to drug interactions. We hope that the cases will help you in your individual practice in the appropriate management of fungal infections.

We encourage you to visit our website www. Fisftrust.org for other resources and contribute to the registry for invasive fungal infections (www.fungireg.in).

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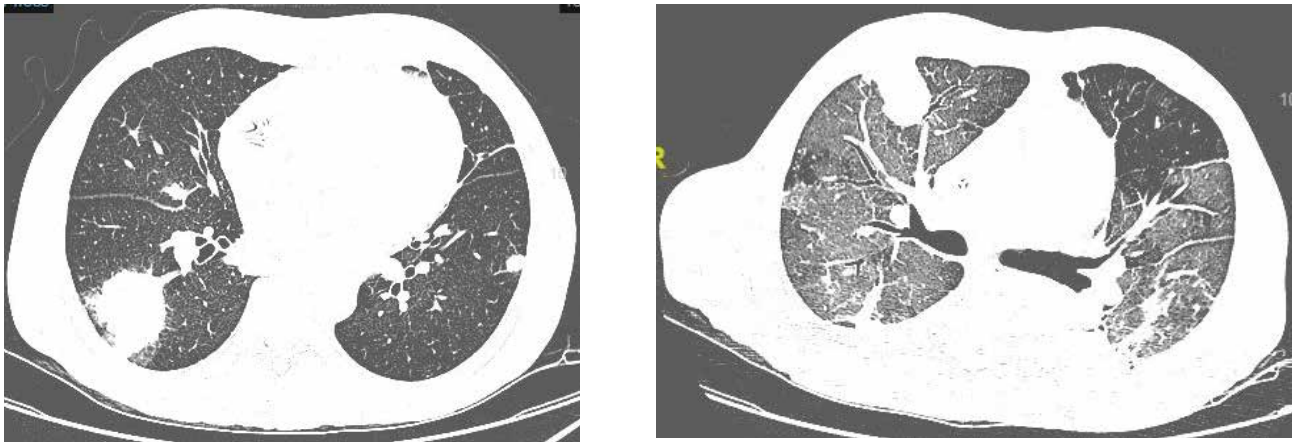

Figure 1: CT chest imaging showing multiple bilateral subpleural nodular opacities with surrounding GGO. (PRETEATMENT)

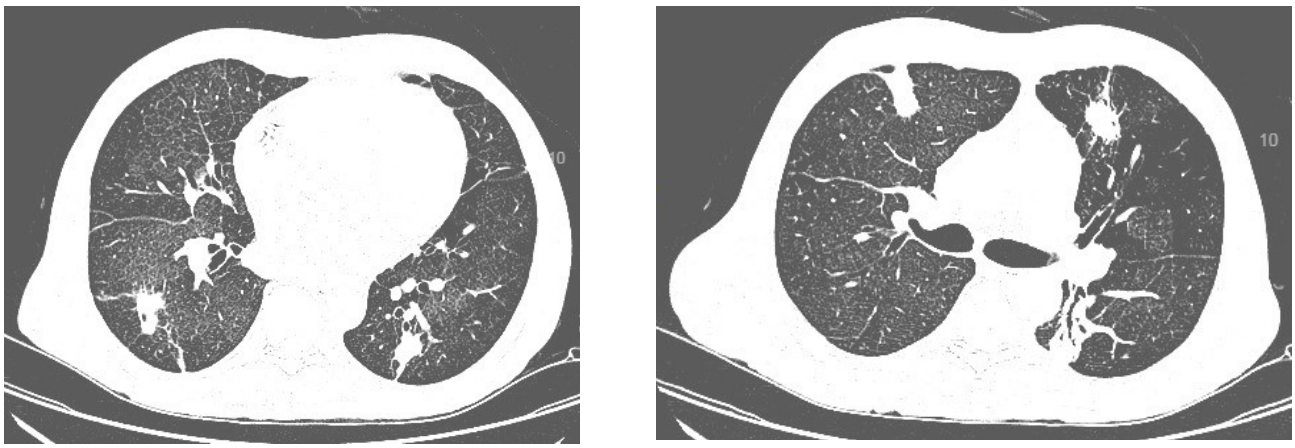

Figure 2: Showing reduction in size of the nodules (3 weeks after treatment initiation)

analysis of the follow up CT scan of the chest taken earlier, although most of the pulmonary lesions had resolved, there was a suspicious nodule in the lower lobe which showed a mild increase in size which could have been due to Nocardia infection. It is also remarkable to note the fact that breakthrough Nocardia infections can happen while on Cotrimoxazole prophylaxis and such cases have been reported in the literature. Patient was initiated on treatment for Nocardia based on AST. Despite aggressive treatment of the pulmonary infection and brain abscess, patient succumbed to illness.

Pulmonary infections following solid organ transplant are of varied etiology. The differential diagnosis that one needs to consider in an SOT recipient with pulmonary opacities includes Mycobacterial infections, Legionella, fungal infections (Cryptococcosis, Endemic mycosis, Invasive Pulmonary Aspergillosis, nonAspergillus hyalohyphomycosis like Fusarium & Scedosporium, Pulmonary Mucormycosis), Nocardia, Rhodococcus ,rarely pneumocystis etc. The etiological agent largely depends on the timeline of infection, graft function, degree of net immunosuppression, rejection episodes requiring augmentation of immunosuppression, prior colonisation, prophylaxis, pre-existing immunity, vaccination status. The importance of detailed history taking and clinical examination cannot be overemphasised in such situations. CT findings in most of the cases provides a clue to the diagnosis. Presence of consolidation most often is seen with bacterial infections while diffuse ground glass opacity is quite often seen in infections like PJP, viral pneumonias. Halo sign (central consolidation surrounded by GGO), reverse Halo sign (central GGO with surrounding consolidation), vascular cut off sign (abrupt termination of pulmonary artery branch feeding the lesion), bird's nest sign are reported in tissue/ angioinvasive mycotic infections. Sending an appropriate respiratory sample (sputum, BAL, transbronchial/ transthoracic biopsy specimen) for microbial analysis is crucial for accurate diagnosis.

This case highlights the following features whenever we encounter an invasive fungal infection in an immunocompromised host:

1. The clinical relevance of isolation of fungi from a non-sterile site needs to be interpreted with caution especially in a susceptible host. This patient met EORTC/ MSG criteria for probable aspergillosis. However, the transbronchial biopsy established the diagnosis as a mixed mold infection. This had treatment implications. If the Mucormycosis would have been missed, it is possible that voriconazole would have been chosen as the antifungal therapy.
2. While in our case, the fungal cultures were positive (though not for Mucorales), failure to grow the fungi on culture plate doesn't necessarily mean an absence of infection. Proper collection, transportation, storage, processing and incubation of appropriate samples are essential to isolate the fungi in the lab. Whenever there is a strong clinical suspicion of an invasive fungal infection, its concern should be conveyed to the microbiologist and pathologist and a careful examination and follow up is essential.
3. Mixed mycotic infection is no longer an unusual entity. With more and more number of cases being reported, microbiologists and pathologists have to be more vigilant in diagnosing such mixed infections as the treatment outcome of fungal infections is largely dependent on the precise identification of the fungi to

species level followed by antifungal susceptibility testing, yet another challenging domain in fungal diagnostics .One can resort to molecular testing methods like ITS (Internal Transcribed Spacer) whenever conventional methods fails to identify the fungus.

4. Preference for a simplistic explanation (as suggested by William of Ockham) does not hold good in a complex situation, especially in a immunocompromised host. In the setting of a pulmonary fungal infection, the CNS lesion could have been attributed to a fungus more so since posaconazole does not have good levels in the CNS. Finding Nocardia as a cause of the CNS disease in our case illustrates the fact that finding one pathogen should not stop one from looking for more, especially when there is treatment failure.

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POST LIVER TRANSPLANT CHOLANGITIS DUE TO CANDIDA NORVEGENSIS: EXTRAPOLATIVE PK PD CONSIDERATIONS FOR TREATMENT

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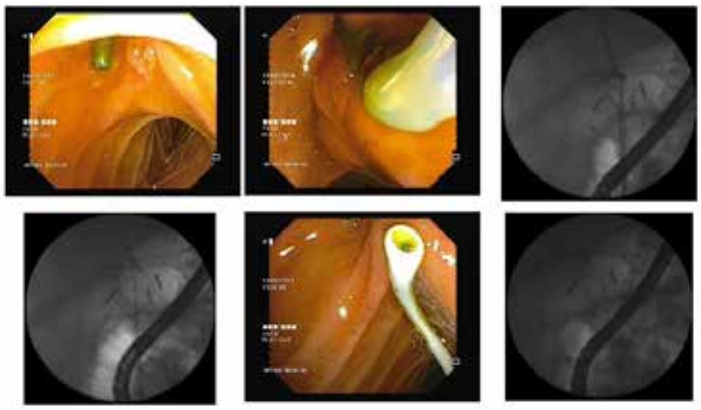
C. norvegensis is an uncommon Candida species causing infection in immunocompromised hosts. It is intrinsically resistant to fluconazole. A strong association with post- liver transplant status, as in this case and near-100% mortality, likely due to inappropriate antifungal therapy and lack of source control has been reported in literature.

Mr. AK, a 32 year old gentleman, 10 months post-liver transplant recipient, had stenting done for a biliary stricture. A month later, he developed ESBL *E. coli* cholangitis and bacteremia for which he was treated with Meropenem. Flaky pus was seen during stent exchange which grew *Candida norvegensis* on culture with 97% probability of identification by VITEK 2 (Figure1).

Suspecting cholangitic abscesses the, patient would require at least 3 weeks of antifungals and Meropenem.

Antifungal susceptibility was done by VITEK since broth microdilution was not available at that time. As there is limited data about antifungal susceptibilities of *C. norvegensis*, MIC's were generated on VITEK by using names of other Candida species eg *C. glabrata* as in Fig 2. Though this is a common practice, it is not fully validated.

Micafungin was found to show an MIC of 0.12 and Voriconazole of 0.25. Since CLSI breakpoints are not available, EUCAST breakpoints were considered. However these are only provided for certain species and extrapolated to *C. norvegensis*. It is interesting to know that while micafungin was susceptible, caspofungin was not. The clinical significance of this differential susceptibility is not known. Anidulafungin



Report:

Duodenoscopy revealed previously placed biliary stent. The stent. The stent showed proximal migration and thre was some pus draining through the stent. The stent was cought in a snare and was removed.

Selective CBD canulation was done. Guide wire was passed deep beyond the biliary anastomosis. Bile sample was atken and sent for C/S

Cholangiogram was taken. It revealed anastomotic stricture.

A double pigtail 7 fr plastic stent was placed well beyond the anstomosis to facilitate bilary drainage.

There was a free of bile and air cholangiogram at the end of procedure.

Figure 1: Endoscopy and Fluoroscopic images

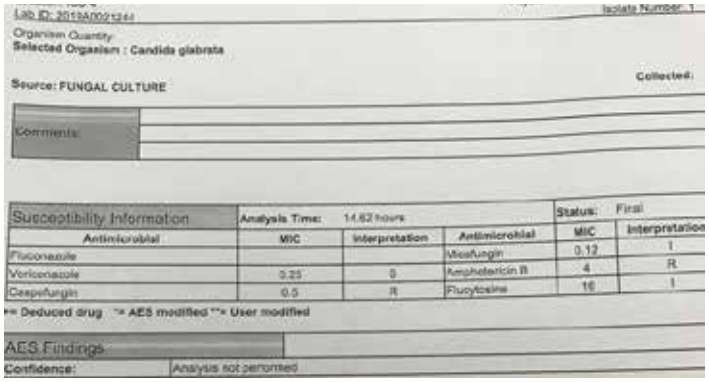


Figure 2: Antifungal susceptibility of the isolate

susceptibility was not done.

The choices for therapy include voriconazole and micafungin. The echinocandins are cidal and have biofilm activity unlike voriconazole which is only fungistatic and has no biofilm activity. There is no data about biliary levels of voriconazole and there is a significant interaction with tacrolimus. The PK PD indices for efficacy of Voriconazole is AUC/MIC of 30 and of Echinocandins is Cmax/MIC of 1. Micafungin dose of 150mg generates a biliary trough level of 1.9 mcg/ml which will lead to Cmax/MIC (1.9/0.12) of 15.83, exceeding the required target Cmax/MIC for cidal therapy of echinocandins which is 1. Hence Micafungin and Meropenem were administered for 3 weeks and the patient responded well to treatment.

This case highlights the importance of speciation of Candida species, extrapolating MICs and Breakpoints for species where data is not available, early source control and use of PK/PD considerations in choosing the appropriate antifungal agent on a case by case basis.

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CRYPTOCOCCAL MENINGITIS PRESENTING FEVER OF UNKNOWN ORIGIN LATE POST KIDNEY TRANSPLANT

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A 35 year old gentleman, Bangladeshi national, presented to us with fever of unknown origin in October 2018 for 4 months. He had successfully undergone a living donor renal transplant (donor- wife) in 2010 at Kolkata. Native kidney disease was unknown. His post-transplant period was relatively uneventful, with triple immunosuppression- tacrolimus, mycophenolate mofetil and prednisolone, which was gradually tapered. There were no immediate post-transplant complications. He has reviewed in June of 2018, when he was noted to have a rise in creatinine from a baseline of 2.8 mg% to 4.45 mg%, at which time graft dysfunction was considered. Steroids were increased without a pulse dosing. He returned for review with fever in October 2018. Detailed work up done at Kolkata was unremarkable, and empiric anti tubercular therapy was instituted in December 2018. This was, however, discontinued after 3 months, as there was no evident benefit. At this point, he was referred to our center for evaluation. He did not report any new symptoms, aside from persistent fever, tiredness and minimal weight loss. Examination was unremarkable.

Baseline labs were done which showed a normochromic normocytic anaemia in an otherwise normal haemogram. Serum creatinine was 4.1 mg% and other labs were unremarkable. Blood and urine culture remained sterile; imaging of chest and abdomen showed no changes; echocardiogram revealed no valvular lesions; PET scan done earlier was reviewed and showed no changes. Rheumatology evaluation was noncontributory.

Evaluation in our department started with a review of symptoms which elicited a history of occasional headaches. On this basis, after reviewing the imaging of the head, a lumbar puncture was performed, which showed a lymphocytic pleocytosis (175 cells, 98% lymphocytes), with a low sugar (24 mg%) and elevated protein (156 mg%). Gram stain was noncontributory, but India Ink stain was positive (Figure 1). Cryptococcal antigen (CrAg) was positive at 1:64 dilution. Cerebrospinal fluid (CSF) culture grew yeast (Figure 1)

Treatment with liposomal amphotericin B and dose adjusted flucytosine was initiated, and after 14 days of therapy, his repeat spinal fluid analysis showed reduction in counts, rise in sugar and negative culture. In the interim, culture was reported a *C. neoformans*, resistant to fluconazole (MIC>16) but susceptible to voriconazole. He was stepped down to oral voriconazole with adjustment of tacrolimus dose, and adequate serum levels were ascertained after a week of therapy. His creatinine also stabilized at 3.5 mg% on follow up.

Although cryptococcal meningitis is typically seen as an opportunistic infection in immune suppressed individuals like those with advanced HIV infection, it presents differently in patients with solid organ transplant recipients. It is more commonly noted in patients in renal transplant recipients than other hosts like liver recipients. Also unlike other transplant associated infections, it presents in the second year after transplant, although it has rarely been noted even later.[1] With wide spread use of calcineurin inhibitors, there is a reduction in disseminated cryptococcal infection, but it still remains a leading yeast pathogen in renal transplant recipients, after Candida.

Diagnosis of disease and estimation of spread is important in management. Blood cultures can be positive in nearly half the patients with neurological involvement, and should be performed. Although CSF examination is the preferred method of diagnosis, with cultures being performed to identify the species, serum and CSF CrAg is the best way to diagnose infection. However, a positive serum test does not predict neurological involvement, making CSF analysis mandatory. Lateral flow assay is an inexpensive and rapid way to diagnose infection, and is positive in the various species causing infection, but is subject to false positivity in low titers and also the prozone phenomenon when infection burden is exceptionally high.

Identification at a species level is now considered standard of care, as it can influence antifungal choices. In fact, rising rates of fluconazole resistance makes therapy challenging, and is often noted in patients who relapse on fluconazole therapy. Raising the dose of fluconazole is often adequate for those infected with strains with higher MIC to fluconazole, but the treatment in situations where the fluconazole MIC is higher than 16 is unclear, and newer azoles can be considered. This has to be coupled with reduction in immune suppression to the extent possible.

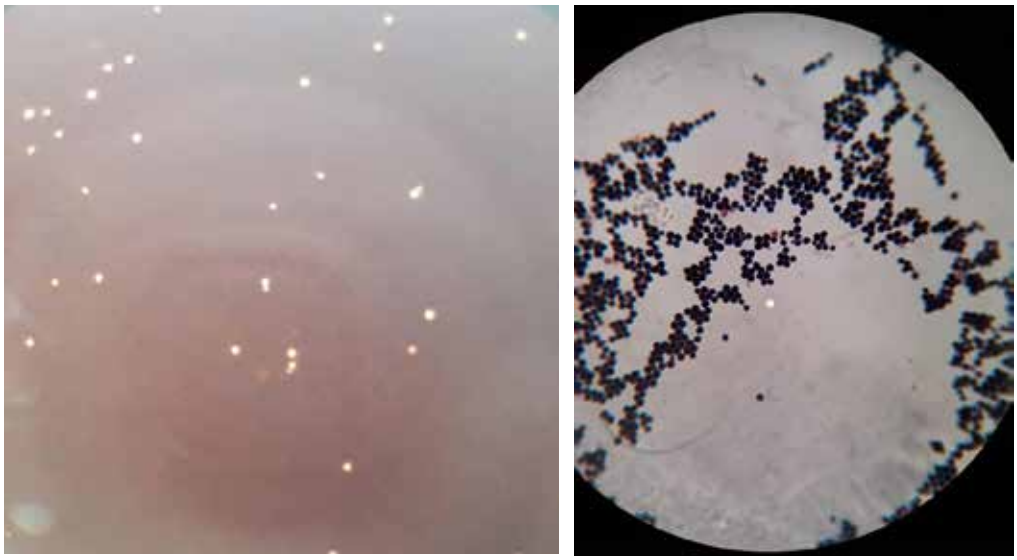

Figure 1: India Ink stain and fungal cultures

In situations where there graft loss, the host should be maintained on haemo dialysis for at least a year, with evaluation to ensure sterilization of infection prior to consideration for retransplantation.

Our patient highlights the concern that cryptococcus can manifest as a very late infection in patients with renal transplant, and the symptoms can be very muted, and requires a high degree of suspicion to diagnose. Prompt identification and therapy has improved the prognosis in such patients.

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FATAL GASTRIC MUCORMYCOSIS IN A LIVER TRANSPLANT RECIPIENT WITH THROMBOTIC MICROANGIOPATHY

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A 53 year old non diabetic, non hypertensive, chronic alcoholic patient with decompensated chronic liver disease underwent live related liver transplant. He received basiliximab and methylprednisolone as induction, followed by Tacrolimus, Mycophenolate Mofetil and Prednisolone for maintenance. Post transplant, he developed thrombotic microangiopathy (TMA) likely related to tacrolimus. It was substituted with everolimus and also given 2 doses of eculizumab and discharged.

A few days later he presented to the emergency room with fever, breathlessness and hematemesis.

The CT abdomen (Figure 1) revealed the presence of hyperdense thickened and heterogenous anterior wall of stomach in the region of body, with intra-mural air locules, along with pneumoperitoneum in the upper abdomen. Upper gastrointestinal endoscopy revealed necrotic stomach wall and blood clots as shown in Figure 2.

The biopsy revealed broad, pauciseptate hyphae with wide angle branching typical of mucormycosis (Figure 3). The histopathology revealed similar hyphae with vascular invasion (Figure 4).

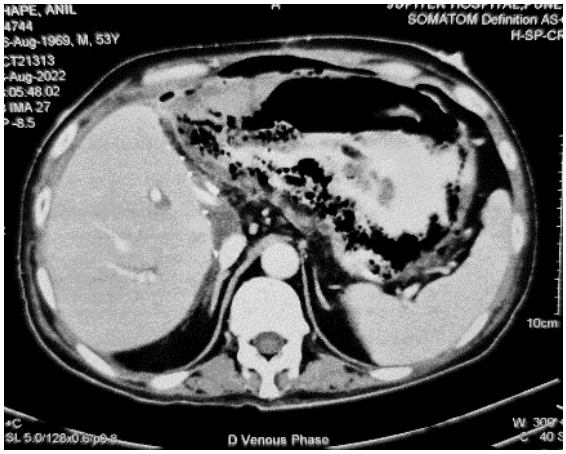

Figure 1: CT abdomen

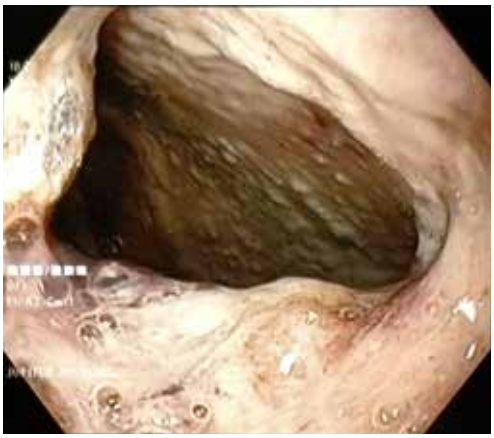

Figure 2: Upper GI endoscopy

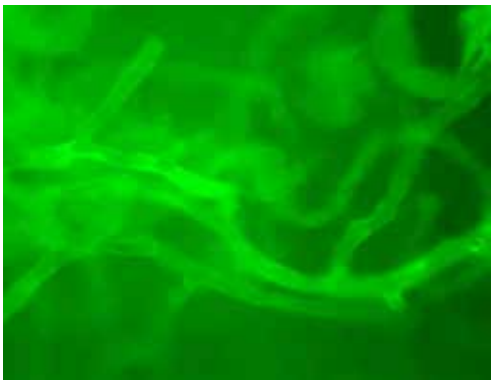

Figure 3

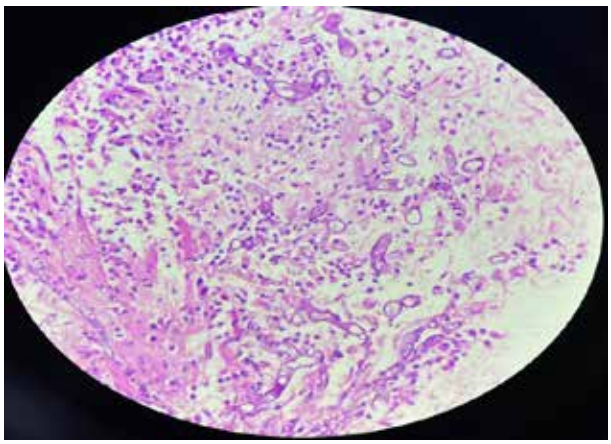

Figure 4

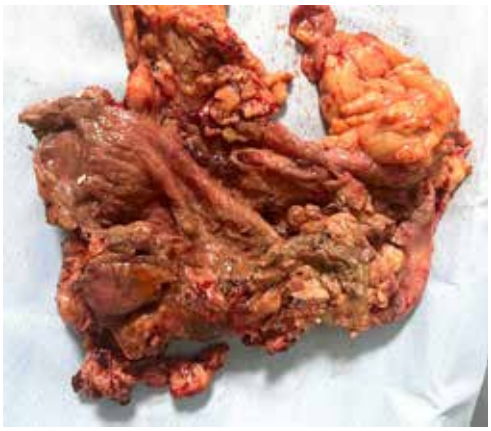

Figure 5

Treatment with IV Isavuconazole was initiated. Liposomal Amphotericin B was not chosen as the patient had developed kidney injury due to unremitting TMA. Gastrectomy, esophago-jejunostomy was done and Amphotericin deoxycholate was instilled locally. The resected specimen is shown in Figure 5.

Despite multiple sessions of plasmapharesis and futher doses of eculizumab, TMA was unremitting and the patient succumbed to multiorgan dysfunction and progression of mucormycosis.

It is plausible that microvascular occlusion due to TMA, mucosal ischemia, severe immunosuppression and ingestion of spores in food may have led to gastric mucormycosis. Progression of disease despite radical surgery and antifungals is possibly due to the fact that the underlying condition did not allow reduction of immunosuppression which is a crucial component of therapy for mucormycosis.

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AN UNUSUAL CAUSE OF THROMBOCYTOPENIA IN A LIVER TRANSPLANT RECIPIENT

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A 54 year old male resident of Indore, Madhya Pradesh underwent living related donor liver transplant in February 2021. The post-transplant period was unremarkable. He was on triple immunosuppression with prednisolone of 5 mg, mycophenolate mofetil and tacrolimus. In November 2021, routine CBC showed a Hb of 12 gm/dl, WBC count of 3400 with 75% polymorphs and a platelet count of 47,000/ µl. He presented in January 2022 with fever and generalized weakness since 1 month. Physical examination showed splenomegaly. The Hb had dropped to 10.5 gm/dl, the WBC count was 1500/ µl and platelet counts were 27,000/µl with an immature platelet fraction of 4.7%. The CT abdomen showed an enlarged spleen (size 18 cm) which had significantly increased in size as compared to the pre-operative evaluation. CMV viral load was negative. The possibility of a hematologic malignancy was considered and the patient underwent a bone marrow aspiration and biopsy. To our surprise the marrow revealed yeast like forms in the macrophages typical of histoplasmosis (Figure 1). No sample had been sent for culture.

Treatment with liposomal amphotericin B was initiated @ 3 mg/ kg/day and continued for 2 weeks. Later treatment was switched to itraconazole loading (200 mg thrice daily for 3 days and then 200 mg twice daily). Tacrolimus dose was reduced and the levels monitored. The itrcaconazole level after 2 weeks was 1 mcg/ml. The patient is doing well on follow up with weight gain, normalization of counts and is still on itraconazole.

The case brings up several points for discussion. Histoplasmosis is under reported in solid organ transplant recipients in India possibly because of poor awareness and misdiagnosis as tuberculosis.[1] The index of

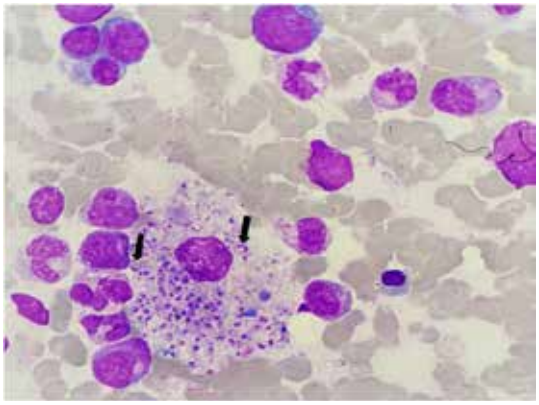

Figure 1: Bone marrow aspirate showing intracellular yeast

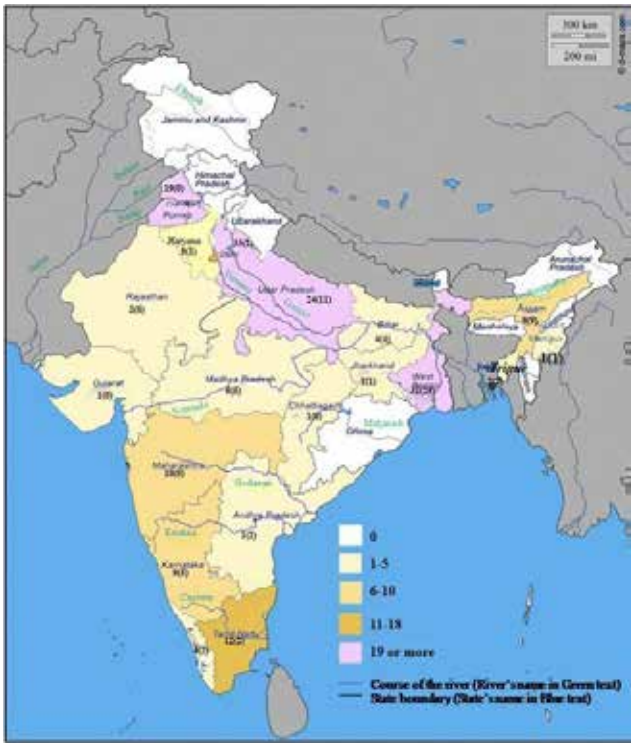

Figure 2: Histoplasma map of India

suspicion is even lower in non-endemic areas as in our patient (Figure 2). However, histoplasmosis is now increasingly being reported from non-endemic areas in India as well.[2] Other pathologic differentials for intracellular yeast like organisms in the Indian setting include cryptococcosis (the yeast are large 4-10 micron as against histoplasma 2-4 micron) and talaromycosis (elongated with clear central septation). There are treatment challenges in the solid organ transplant setting due to interactions between itraconazole and tacrolimus. Finally the duration of therapy is not well defined. Literature suggests that treatment can be stopped after 12 months and patients monitored for relapse by serial urine histoplasma testing.[3] The availability of urine histoplasma antigen test is a limiting factor in our setting.

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REMOTE INOCULATION MYCOSIS: RIP VAN WINKLE WAKES UP

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A remotely inoculated organism may be activated to produce clinical disease following immunosuppression. We describe such a case with infection due to *Medicopsis romeroi,* a rare mould.

This is the case of an 54 Y/M, diabetic, hypertensive, status post living related donor renal transplantation, done in February 2021. The patient was on standard triple immunosuppressant regimen. He developed a painless nodule on his thumb over 5 months which did not respond to multiple courses of antibiotics. The swelling was excised and sent for various tests. Review of USG after Infectious disease referral, showed a small foreign body, like a wooden splinter in the wall of the lesion (Figure 1). On enquiry, a 3 mm wooden splinter in the lesion had been noted at surgery and the patient recalled an injury at the same site, 20 years ago when he used to work on a farm. Organisms derived from soil or thorn injury including bacterial and fungal organisms were considered in the differential diagnosis. Bacterial organisms were considered less likely as there had been no response to antibiotics.

Histopathology showed brownish septate hyphae with constrictions at the areas of septations (Figure 2). The excised tissue grew a dematiaceous mould (Figure 3). In Lactophenol Cotton Blue (LPCB) mount branched, septate hyphae with sparse conidia (Figure 4) were seen. MALDI- TOF MS was unable to identify the mould. Sequencing identified it as *Medicopsis romeroi.* There are no Epidemiologic cutoffs (ECOFFs) or break points (BP) available for *Medicopsis romeroi.* Minimum inhibitory concentration (MIC) of Voriconazole (VCZ) is reportedly low and hence was chosen for treatment with appropriate dose adjustment of tacrolimus.

This case is aptly likened to "Rip Van Winkle" an American folktale character who returned to his village after a 20 year sleep in the woods. This case underscores that remote inoculation, when the patient was immunocompetent, could have introduced a mould, which remained latent and reactivated after immunosuppression. Sending excised tissue for appropriate tests is incredibly rewarding. The case also illustrates the need for sending an isolate to a reference laboratory for identification if standard methods are not able to identify the pathogen. *Medicopsis romeroi* is a rare mould with only 12 cases reported so far. It is difficult to identify except with sequencing. There is no standard guidance on treatment. Surgical excision along with prolonged treatment (at least 3 months) with one of the new azoles is recommended.

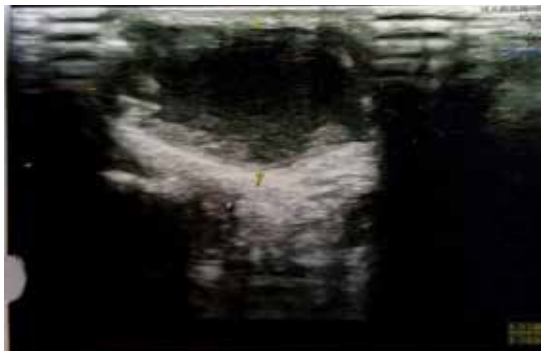

Figure 1: USG showed a small foreign body in the wall of the lesion

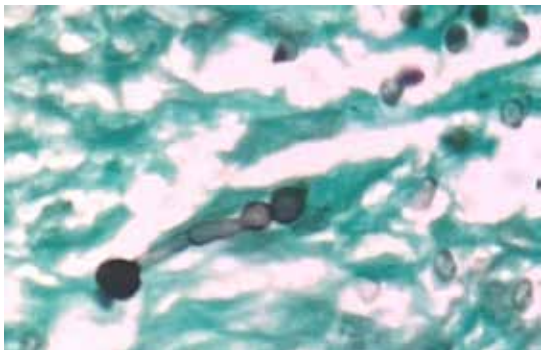

Figure 2: Histopathology showed brownish septate hyphae with constrictions at the areas of septation

Figure 3: Excised tissue Grew a grey to black mould

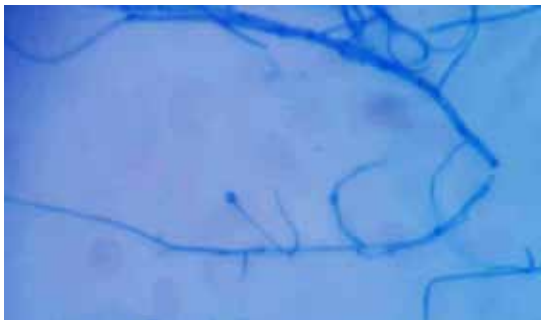

Figure 4: In Lactophenol Cotton Blue (LPCB) mount showed branched, septate hyphae with sparse conidia

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About FISF

The purpose of the Fungal Infections Study Forum is to conduct educational activities, undertake epidemiological and clinical studies and to promote research activities on invasive fungal infections. The results of such research would benefit the clinicians, mycologists and the general public. The trust was formed in view of emergence of Invasive fungal infections (IFIs) in India which is posing a serious challenge to the haematologists, critical care providers, ID specialists, pulmonologists, neurologists, medical mycologists and many other clinicians handling serious and immunocompromised patients. The trust is the independent working consisting of clinicians and mycologists and instituted on 3rd March 2012 at Mumbai, India. To know more about us visit www.fisftrust.org.

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